CONTRIBUTIONS
TO
CARDIAC PATHOLOGY

DR. COCKLE.

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BY

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PREFACE.

The following papers, scattered through the journals, are here collected, with a few notes and alterations.

Some questions they more or less incidentally discussed, would seem yet to await a final answer. For example, whether all the elements are found for a thoroughly satisfactory explanation of the first sound of the heart; the precise period of cardiac revolution, during which injection of the coronary arteries takes place; whether *data* are furnished for possible diagnosis in transposition of the great vessels of the heart; whether presystolic murmur may not, at times, be of indirect or regurgitant origin; and lastly, the immediate cause of death in insufficiency of the aortic valves.

The signs revealed by auscultation, in some cases, still admit of more correct interpretation.

As Professor Germain Sèe has well remarked in a recent work*—'Well then! Do not deceive yourselves. The subject is far from being exhausted. It were a strange delusion, did you expect to find at the bed-side the living reproduction of the picture, apparently so clear and simple, which is traced in books. Clinical observation will daily show you both the number and extent of the lacunæ that remain to be filled in the history of diseases of the heart; and it is while bringing new and important matter to this vast pathological edifice, that you will, at the same time, be made sensible of its fragility.'

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* *Du Diagnostic et du Traitement des Maladies du Cœur.* Paris, 1879, p. 1.

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ON THE SECOND SOUND AND MURMUR OF THE HEART AND GREAT VESSELS, IN THEIR RELATION TO DIAGNOSIS.*

The following contribution has for its object an inquiry into the second sound and murmur of the heart and great vessels, in their relation to diagnosis. It is solely one of practical character; in some points, not altogether in accordance with the current teaching; and is, consequently, open to the fullest discussion and criticism. The facts are stated as they occurred to me, together with such inferences as may be considered fairly warranted from the subject matter.

I shall premise a few observations generally respecting the impulse, rhythm, and sounds of the heart, before entering into the question of the special modifications in disease connected with the second sound, etc. The ordinary expression of the heart striking against the chest-wall appears inaccurate, since it is now pretty generally admitted that the heart, in the perfectly normal state, never disconnects itself from the parietes. The conditions necessary for an actual stroke are, therefore, absent. In the language of Charles Robin, 'On ne peut frapper ce qu'on touche, et l'on ne bat que ce que l'on ne touche pas'. The apparent stroke consequently appears to consist simply in elevation of the corresponding soft structures of the chest-wall.* This matter, however, is too complex to challenge from me here the arguments in its support. To pass on, then, to the rhythmic action and sounds of the heart, I may state that I have for some time past been engaged in an experimental investigation of these phenomena. Taking into consideration the retrograde course of the coronary arteries, and the fact that, during the systole of the ventricles, the blood of the coronary arteries would be prevented penetrating their tissue, it occurred to me to revive the old inquiry as to the time when the nutrient arteries of the heart were filled. I remembered the physical demonstration of Bernouilli, that, if fluid was injected into a tube of given calibre, it would pass with facility into secondary tubes, the direction of which was in harmony with that of the main branch; but if, on the contrary, the direction of the secondary tube was opposed to that of the trunk, not only would the onward current be prevented passing into such diverticulum, but that fluid would actually be drawn up into the latter from a reservoir below.

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* *British Medical Journal*, 1857.

I applied, I say, at first, this argument to the action of the coronary arteries, and conjectured, from their direction with respect to the main trunk, that they must be filled during aortic systole. Subsequently I found, experimentally, that this chain of reasoning, however sound with regard to fluid traversing empty tubes, would not accurately apply to those already filled. I have consequently thus modified my view with respect to these phenomena.

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* What is ordinarily termed the apex beat is, probably, at least during full inspiration, but the sudden hardening of a portion of the right ventricle.

While admitting fully a controlling influence of the nervous ganglia of the heart, I adopt, at the risk of being thought antiquated in my notions, the Hallerian doctrine relative to the automatic action of this organ; and contend that, with reference to the invariable sequence between the systole and the diastole, it is primarily one of contractility, conditioned upon the contact of the blood with its tissue.

I conceive that the heart's alternating systolic and diastolic action is, in the healthy subject, maintained by two causes; the one intrinsic—the contact of blood with the endocardial membrane; the other extrinsic—the blood injected by the coronary arteries into the proper tissue of the muscle. The necessity for the first cause—the ingress of blood from the cavæ into the auricle—seems proved by the early experiments of Bartholini, and their subsequent modification by Haller, Cardan, Senac, and others.* The second cause I attempt to explain in the following manner, commencing from that period of a revolution of the heart's rhythm corresponding to the ventricular systole. At this time the auricle is dilating from the blood of the cavæ; its tissue is lax, and easily permeated by aortic arterial blood. These two acts, being simultaneous, determine at once its contraction. The contracted condition of the ventricle during this period had prevented the entrance into its tissue of the nutrient blood; the pallor of the heart's surface is most marked; indeed, in some of the lower animals, the walls appear almost transparent; but now, commencing its diastole, and filling with the blood from the auricle, the obstacle is removed, and instant contraction again ensues, as before described.

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* For a brief notice of these experiments, see a short note by the Author, *British Medical Journal*, January 31, 1857.

With regard to the sounds originating at the cardiac orifices, I accept the teaching of Skoda, that there are two engendered at each venous, and two at each arterial ostium.

I incline, however, to the belief that the first ventricular sound may have an accessory element of causation as follows. During the closure of the auriculo-ventricular valves, they carry, in the funnel shape partially assumed during the process, a portion of the ventricular blood back into the auricles: this portion is reinforced by blood streaming from the cavæ and pulmonary veins; so that the reaction between the lower and upper columns, the valves intervening, contributes, to some extent, to the formation of the dull, prolonged, first ventricular sound, the upper blood-column acting as a damper to the valve sound. And this view leads me to consider that, during the ventricular systole, the chamber cannot be entirely emptied.

If, during the period of ventricular contraction, we imagine the blood-stream divided into two columns, one rushing through the aortic mouth, the other assisting in the momentarily sustained closure of the auriculo-ventricular valves, this latter portion must, theoretically, by the nature of the contraction, be left in contact with the ventricular face of those valves, and rendering their mid portions of dome-like form, which latter condition has been recently experimentally demonstrated by Chauveau.

However this may be, one point appears clear, if the recent experiments of Dr. Halford may be considered trustworthy, viz., that the obstruction of the blood from the cavæ into the heart annihilates the normal sounds. Some indefinite sound, it appears, may still exist; muscular bruit, the visible vibrating motion described by Haller during the contraction, caused possibly by the stimulus of the blood, still existing in the coronary arteries, upon the muscular tissue of the heart. Hence we may exclude the notion of the first sound, at all events, being principally muscular.

Of the several theories proposed in explanation of the second ventricular sound, that of Julius Roger appears to me the simplest and the best—viz., that membranes may cause sound both by impact and by separation. The wedge-like column of blood traversing the auriculo-ventricular orifices may, by impinging upon the separating valves, engender sound, a certain amount of force being imparted to the blood-stream, to prevent the tendency to partial vacuum left by the relaxing ventricle. Physiologists are not clear as to the actual amount of overlapping, but it is doubtless sufficient to explain the phenomenon. And one fundamental fact is admitted by most auscultators—viz., the occasional occurrence, in the normal state, of a second sound highly intoned at the apex, while the basic sounds are weak, and, it may be, of different pitch. I shall advert to this point again, when speaking of murmurs at the apex.

The second arterial sounds are supposed to have their *maxima* at or near the mid sternum, and, for purposes of differential distinction, are recommended to be auscultated at the aortic and pulmonic cartilages respectively. Whether the information sought be gained by this procedure, has always appeared to me doubtful. To solve this point, I have recently attempted various experiments. The following was one. Two watches, of nearly equal timbre, intensity and synchroneity, were covered with oilskin bags, and to the neck of each an India-rubber tube was attached; the watches were placed one upon the other; and the tubes, after being connected at their origin, were made to diverge to a given distance. It was always impossible, upon listening alternately at the open end of each tube, to distinguish the relative position of the watches, the mean note only being heard. They were afterwards introduced into the aorta and pulmonary artery respectively of an ox recently slaughtered.

Auscultation, as before, always yielded the same result. Media of different density, brought temporarily into contact with the free portion of a tube, modified the note at its open end.

It must, however, be borne in mind that synchronous with those arterial sounds, is the sudden injection of blood into the auricles from the large veins; and, from their unequivocal muscularity, it is hardly possible to conceive that this impulse can be altogether aphonous. According to a celebrated physiologist and auscultator of a certain school in Paris, Dr. Beau, although in a different sense, the auricles are said to yield under these conditions distinctly audible sound.

With respect to the second arterial sounds, it is taught by some, that the aortic sound is loudest; and this would, certainly, on *à priori* grounds, be probable, considering the area of the vessel and the stronger valves at its mouth. So far, however, as my own observations extend, I have not found this assertion practically true. I have observations relative to this point extending to one hundred cases, and I found on analysis they gave the following results.

About 60 per cent. equal at both aortic and pulmonic cartilages.
,, 26 ,, loudest at aortic cartilage.
,, 14 ,, loudest at pulmonic cartilage.

The question may therefore fairly be asked, whether the loudness of the second sound does stand in a direct causal relation to the strength of the ventricular systole and the reaction of the arterial walls?

I have observed in typhus fever, fatty heart, spanæmic women, and cases of general debility, that when the first sound was decidedly weak, the second was still loud and clear. In the so-called accentuation of the second sound of the pulmonary artery where it would be said to exist, it has appeared to me usually rather a forcible recoil of the artery than an accent properly so called.

Taking the second aortic sound, or its replacement by murmur, as a basis for differential diagnosis, the following points have presented themselves to my notice.

A preternatural accent of the second sound in the direct course of the aorta, to the right of the sternum, has been observed and specially indicated in some cases of aortic aneurism. I have recently had an opportunity of watching throughout a case of aneurism of the ascending aorta. The earliest symptoms were those which apparently indicated an inflammatory condition of the aorta: at this period, the only persistent physical sign appreciable was marked intensification of the second sound over the aorta. Some months later, pulsating tumour was seen and felt about the second and third right costal cartilages; the second sound still being preternaturally loud. In the early stage, it occurred to me that the loud second sound might be explained on the hypothesis that the inflammation of the aorta had induced unusual contractility of this vessel, and thus, by rendering it more rigid, making it a better conductor of sound. At a later period, the extensive thickening of the coats, by excessive deposit, might have rendered the tube a better conductor; at all events, the intensified sound could not be explained upon the hypothesis of increased recoil of the vessel, since here the elastic reaction of the vessel was impaired or even destroyed by extreme degeneration. A further argument against the loudness of the second sound being supposed to result from the injecting force of the heart, may be borrowed from Dr. Stokes. When treating of softening of the heart in typhus fever, he states, that in his 41st and 56th cases, the second sound was preternaturally loud, and in the latter case attended with impulse, while the first sound was extinct, and the impulse not appreciable by palpation. I again submit, therefore, that the hypothesis of the causal relation between the loudness of the second sound and the propulsive force of the ventricle, requires reconsideration.

It is hardly necessary to mention induration of the pulmonary tissue, excavations therein, contraction of the lung from phthisis, permitting the pulmonary artery to come in immediate proximity with the chest wall—a pathologic condition first indicated, I believe, by Dr. Sibson—enlargement of the liver, etc., as sources of intensification of the sounds. I have, however, some reason to believe that a marked intensification of the sound in the post clavicular space may, now and then, assist in the diagnosis of deposit at the apices of the lungs.

In insufficiency of the aortic orifice, the second sound is more or less perfectly replaced by murmur, soft, harsh, or musical. Such murmur, however, does not nor cannot measure the amount of patency. I have particularly observed that in some cases, in which, judging from the extraordinary collapse of the arteries, the patency was extreme, that the murmur in its physical characters differed in no respect from other less advanced cases in which no collapse was observed.

I believe enlarged experience scarcely supports the notion of Dr. Hope, that aortic may be distinguished from other murmurs by the pitch. The pitch of a murmur at any orifice infinitely varies with the physical condition engendering it. An aortic regurgitant murmur may be of so high a pitch as to approach sibilation in point of intensity.

I may add, that this distinguished observer was also decidedly in error when he stated that a mitral murmur was never audible above the level of the aortic valves. Murmurs at the orifices are not so restricted as many suppose.

There are one or two points, however, with regard to aortic diastolic murmur, which I will take leave to mention. The first is that, contrary to the opinion of some observers, I have not unfrequently found it attended with well marked diastolic fremitus even at the apex, and this in cases unattended with any excessive degree of spanæmia; and, indeed, there appears no reason why a partially rigid valve should not be susceptible of being thrown into tactile as well as sonorous vibration by the refluent current.

It is usually taught that the differential diagnosis of aortic regurgitant murmur may be, at least, in part, established by the clear click of the valves of the pulmonary artery auscultated to the left. I am far from asserting this is not occasionally correct; but so far as my opportunities of observation extend, I should, in the majority of cases, deny the validity of this proposition.

When the diastolic murmur is at all intense, I believe the second sound of the pulmonary artery is always more or less obscured, and in musical murmur completely so. Indeed, if we bear in mind the anatomical condition existent, and consider how readily vibrations are excited in contiguous and homogeneous structures, the difficulty of conceiving the reverse is obvious.

The limits of these murmurs again, and particularly the musical, have been too much restricted; they extend over a very wide area: at times, all over the anterior and posterior surfaces of the chest; from the epigastrium to the pubis; and posteriorly, from the vertex nearly down to the lower end of the sacrum. From their site of origin, they may, I am quite sure, from comparison of cases examined after death, extend across the apex and round the axilla to the posterior scapular region; and this without coexistent disease of the mitral valve.

Excluding from consideration some rare cases of aortic aneurism, described by Gendrin as being liable to be confounded with aortic patency, is there any other condition which gives rise to similar physical phenomena? I believe there is.

A few months ago, I exhibited, to the Pathological Society, a specimen of diseased suprarenal capsules; they were removed from a female advanced in life, who had exhibited well marked symptoms of cardiac disease: a harsh double murmur was heard, with its maximum near the base, and extending with great intensity up the aorta, and downwards to the apex, axilla, and posterior scapular region. The heart's action was somewhat heaving, and its rhythm irregular. These phenomena were persistent, and scarcely underwent any change when examined on several occasions by Mr. Robinson (whose patient she was) and myself. We considered there might possibly exist both aortic and mitral disease. After death, hypertrophy of the heart was found. The aortic and mitral valves were apparently intact; but the aorta, at its arch, was nearly converted into a bony tube; and in its ascending portion, rough, calcified portions projected, the lining membrane being entirely destroyed.

In another case, of Dr. Hawksley's, similar physical phenomena were constantly observed, and the same irregular rhythm. After death, enlargement of the heart was found; rough, calcified projections studded the ascending aorta and its arch; a slight calcification was certainly found at the base of one of the aortic valves, but not preventing tolerably fair closure; and, to the best of my judgment, quite inadequate to the production of the intense double murmur audible over base, apex, and axilla.

I pass now to the consideration of the second sound and murmur of the pulmonary artery. It has been already mentioned, that any change which places the artery in more immediate contact with the chest wall—contraction of the left lung, for example—allows its pulsation to be both seen and felt, and unequivocally intensifies its sounds; excavations in the superior and middle lobes of the lung; induration of its anterior border, by immediate pressure upon the artery, modify the sounds.

The following case strikingly illustrates such change from this latter condition. Jane Barnes, aged thirty, married, of short stature and florid complexion, was admitted a patient of the City Dispensary on December 11th, 1856. She attributes her illness to sleeping in a damp bed six years ago. Since that time she has suffered from dyspnœa and cough, with yellow expectoration. At present, in addition to these symptoms, she complains of pain in the shoulder blades, the right more particularly; profuse nocturnal perspiration; very slight loss of flesh; she lies most easily upon her right side. She has not menstruated during the last two years. She has never had hæmoptysis. Her mother is still living; but consumption has occurred in some members of her family.

Inspection.—The chest is deficient in symmetry. The left scapula is nearer the spine, and not nearly so mobile on inspiration as the right scapula. The apex of the heart is nearly *in situ.* The fingers are clubbed. She has slight lividity of lips.

Palpation.—A silvery *fremitus* is felt over the second and third left costal cartilages. Pulse weak; slightly quickened, but quite regular.

Percussion.—There is marked dulness and sense of tactile resistance from below the left cavicle to the mamma. A somewhat tubular note is heard under the right clavicle.

Auscultation.—Under the right clavicle are heard phenomena of cavern or dilated tubes. Under the left clavicle, bronchial breathing; the respiratory murmur is absent to the mamma. Between the second and third left costal cartilages, both sounds of the heart are entirely replaced by hoarse murmurs of nearly equal length and intensity; loudest during expiration. To the right of the sternum, the sounds gradually acquire the normal character, as also in the carotids and at the apex.

Dr. Elliotson, who examined this case, made the interesting observation that, upon placing the patient in the recumbent position, the first murmur became converted into leather creak sound.

It is stated also, upon the highest authority, that insufficiency of the mitral valve, *par excellence*, induces marked intensification of the second sound of the pulmonary artery. That such result may occasionally obtain, seems admitted by most observers; but, even at the risk of being considered thoroughly in error, I must express my total dissent from the universality of this proposition. Of course, if it be assumed that insufficiency of the left venous valve cannot exist without such accent characterising it, I have no argument to offer; as *post mortem* examinations, made of cases with the observed sign, do not very often present themselves to my notice. Besides, I have already attempted to show that accented loudness of the second sound does not necessarily depend upon the force of the ventricular contraction; also, that the murmur accompanying or replacing the first sound, often masks more or less the second. I can only say, I have made many examinations after death where the mitral valve had unquestionably allowed free regurgitation, and the sign before death did not exist in any marked degree; and again, in a large number of cases observed upon the living, with all the other physical signs, save increased accent, supposed to characterise such change, we are forced to the conclusion, that such signs always indicate some intraventricular change, other than regurgitation, a consequence, I fancy, opposed to our diagnostic convictions. In support of the views advanced, I think I may cite a high authority, Dr. Walshe.

In extreme *stenosis* of the aortic orifice, as Kürschner has most truly observed, the chambers of the heart and pulmonic system are also kept unnaturally loaded, and would, theoretically, tend equally to a more marked accent of the second sound of the pulmonary artery.

In marked aortic, as in mitral valvular disease, however, it appears to me that we often render ourselves liable to the charge of confounding relative with absolute increase of sound of the semilunar valves of the pulmonary artery. In both instances, the aorta, receiving a diminished supply of blood, would have its valvular sound of closure weakened by want of the requisite distention, leaving that of the pulmonary artery in relative excess.

I would also hazard the passing remark, that I believe slight mitral regurgitant murmur to be extremely common, and that it neither necessarily intensifies the second sound of the pulmonary artery, nor developes any marked dilated hypertrophy of the chambers of the heart.

That congenital malformation of the valvular apparatus of the pulmonary artery, and, by inference, a patent condition of the *foramen ovale,* may also cause the second sound to be entirely masked by a prolonged systolic murmur, has, I conceive, a high degree of probability in its favour. I specially refer to a case published by me in the *Association Medical Journal* for September 20th, 1856, and which has been reprinted as a supplement to the present paper. Both the cases of pulmonary arterial murmur alluded to have been examined by many of the most distinguished observers in this city.

I must, however, leave the subject in other and abler hands, and content myself with expressing the conviction, that, ere long, physical phenomena characterising a transient or persistent alteration of the valvular mechanism of the right side of the heart will be found less rare than has been hitherto imagined; although naturally bearing a very small proportion to those of the left orifices.

In organic changes of the left auriculo-ventricular orifice, the second sound may undergo modifications with regard to its intonation, or be replaced altogether by murmur.

First, with regard to the alteration of the sound in mitral regurgitant disease; it may be either annihilated or intensified at the apex; and how are such opposite conditions to be explained? So far as relates to the occasional extinction of the second sound at the apex, the explanation in some cases is not so difficult. A prolonged (and it may be) harsh systolic murmur, with its maximum near the apex, may entirely replace the first normal sound, and usurp all that period of a revolution of the heart's action which should be filled up by the periods of silence and second sound of the heart, and permit but a barely appreciable cessation before the succeeding systole; indeed, almost the only knowledge we possess that the murmur is not absolutely continuous, is, rather by the distinction between the decrease and increase of the sound; or, at other times, the second sound may, upon very careful auscultation, be faintly heard through the murmur. In such a case, as may be readily shewn by experiment, a blowing sound of intense pitch will mask a sound of lower pitch in its neighbourhood; so, in this instance, the second arterial sound is more or less drowned amid the intense and prolonged whirl of the murmur, but the sound may be frequently again audible at a point remote from the focus of the murmur. Another cause may probably concur in weakening the second arterial sound. Should a murmur be very prolonged, and, it may happen, the regurgitation considerable, we may assume that the left auricle would be kept preternaturally distended. Under these circumstances, the overcharged auricle would press upon the origin of the aorta, and even the pulmonary artery, and by such pressure tend to weaken the natural sounds of closure; and, also, of itself tend to produce murmur by pressure on the vessels at their origin. At least, this is the only explanation to which I could come when listening for the accented pulmonic sound in some supposed cases of mitral insufficiency with congestion of the pulmonic system, when the second sound was no longer audible at the apex. There is another cause, also, capable of damping sounds, especially the second; and that is, excessive accumulation of fat in the neighbourhood of the base of the heart. When, on the contrary, the second sound is distinctly audible at the apex, I think, generally, the systolic whiff is not prolonged, nor the auricle overcharged; so that the second arterial sound may be transmitted from the base.

But the difficulty is not altogether met even here, for, occasionally, the second sound is heard of different pitch, and louder, at the apex, than at the base; and in these cases the argument is strong for the origin of this sound in the neighbourhood of the mitral orifice. It is clear that the theory of Roger would not apply here; because, if the insufficiency were caused by the valves not approximating, they could engender no sound from actual separation, but still a conceivable source of second sound is obvious: the over-filled and, perchance, thickened auricle would naturally discharge its contents, in part, into the left chamber immediately upon the cessation of the systolic act, and the blood, impinging upon valves more or less thickened and rigid, might give rise to the sound in question.

I now pass to the replacement of the second arterial and ventricular sounds by murmur, over the body and apex of the ventricles.

The occurrence of such murmur, engendered at the right side of the heart, is, avowedly, so rare, that it scarcely admits even a passing mention. I am not aware that a well detailed case exists, and yet the conditions for the production of murmur undoubtedly occur now and then. The best recorded instances in which such special changes occurred, limited to the right side of the heart, were discovered prior to the invention of the stethoscope; I allude to the remarkable cases of Morgagni, Bertin, etc. With regard to the comparatively common constrictive disease of the mitral orifice, the most extraordinary discrepancy of opinion prevails; indeed, if there be one point in the history of physical cardiac diagnosis, which, even in the present day, requires, more than any other, an exact comparison of the auscultatory phenomena with the *post mortem* change, it is this, beyond all doubt. To illustrate my meaning more particularly, I will quote the opinions of some great authorities. Skoda states, that, in auriculo-ventricular contraction, "the second sound in the left ventricle is replaced by a murmur, often so prolonged as to be interrupted only momentarily during the heart's systole". Barth and Roger, although admitting that a murmur in lieu of the second sound may characterise the lesion in question, yet state that, in some cases, they have heard a *bruit* during systole alone. On the other hand, Fauvel, a highly distinguished observer (*Archives Générales*), after most careful inquiry, challenges the production of a single well recorded example of abnormal bruit replacing the second sound at the heart's apex coinciding with auriculo-ventricular contraction. He has published some well marked cases of such change, the physical signs co-existing with which were most faithfully noted during life, and compared with the physical changes ascertained after death; in all, without exception, the murmur was presystolic or systolic.

Now how are such diametrically opposed statements to be reconciled, each party appealing to a positive experience? So far as my own opportunities permit me to judge, I should say, without denying the counterstatement, that Fauvel's opinion contains most of positive value: at the same time, I am ready to admit, that to distinguish between presystolic and post-diastolic murmur is, practically, of immense difficulty. The important point for us, however, as physical explorers, is to ascertain the time of the murmur in contracted mitral orifice, that is, whether it most commonly attends the systole or diastole, or both. The difficulty is occasionally great, from coexistent lesion of the aortic orifice.

With regard to the differential diagnosis of aortic and mitral valvular disease, both constrictive and regurgitant, it is of importance to ascertain the character of the sounds in the carotid arteries, and to determine, by percussion, whether enlargement of the heart preponderate in the oblique or transverse diameter. The necessity of this latter knowledge has been pointedly enforced of late years by writers, both of the German and French schools; the merit, however, of systematically directing attention to this pathologic change, at least in the dead body, is due to that distinguished pathologist, Dr. Adams of Dublin. His Essay is a model of investigation, and deserves the most careful study of all who seek a philosophical explanation of the mechanism of the disorders of the circulation.

I have known contracted mitral orifice often to run a fatal course, without other physical evidence than deranged rhythm of the heart, and constant irregularity of the pulse. The heart's sounds in other cases, and perhaps the most numerous class, acquire the character which Skoda designates as "indeterminate", associated with a murmur, faint and circumscribed, extremely difficult to indicate, fusing into systole, diastole, and periods of rest. There is one condition which might, on the ordinary theory, be mistaken for auriculo-ventricular insufficiency and contraction; namely, calcification of the aorta. In this affection, double murmur, more or less intense, may be heard, even at the apex. The rhythm of the heart is greatly disturbed; the contractions very unequal: the pulse small, quick, weak, irregular; but the differential diagnosis may, I think, be determined by the fact of the heart's area being generally, in aortic disease, increased in its oblique diameter; by the murmur being more intense, and traceable over and up the aorta, and certainly not lessening in its ascent.

In a case of suspected adhesion of the pericardium to the left costal pleura, I particularly observed that the second sound at the apex was converted into one of a dull, jogging character.

I have, finally, briefly to allude to the reduplication of the second sound of the heart, both at the base and of the apex, in so far as it may subserve our subject of diagnosis. Indeed, I think this very common phenomenon deserving a far greater attention than it has hitherto attracted. In a paper published by me in *Association Medical Journal* for May 24 and 31, 1856, I detailed some cases of interest; since then I have met with the following cases, in which the phenomenon was unusually marked.

I recently saw, with Mr. W. B. Whitfield, a patient who was suffering from cardiac and intercurrent pulmonary disease. Mr. Whitfield had been struck with the singular repeat of the second sound at the left apex, and made particular mention of this fact to me before I examined the case. The first and second sounds at the base were more or less masked by a short, hoarse *souffle*, and the rhythm of the heart was much disturbed. At the left apex the first sound was still somewhat obscure, but the second was loudly reduplicate without a trace of murmur: the phenomenon had persisted some considerable time.

In a second case, the patient, a young woman, was evidently phthisical; she had recently suffered from violent hæmoptysis; at the base, the second sounds were loudly and distinctly reduplicate; while, at the apex, the second sound was heard clear, single, and defined.

The last case I have to mention, and which has been examined by many competent observers, is so unusual, that I think it right to state it in detail.

Case. Alfred Adams, aged 24, a coppersmith by trade, of middle stature, slightly built, of dark brown skin and florid lips, had an attack of hæmoptysis five or six years ago. A second attack occurred eight months ago; he thinks a quart of blood was expectorated altogether in this last attack. It occurred at night, and was preceded by cough.

Present symptoms. He has dyspnœa; cardiac and epigastric pain, of burning character on exertion; pain in the arms; palpitation, which has existed for some years; cough, with slight yellow expectoration; great nervousness; morning sickness; he sleeps in the horizontal posture. He has never had rheumatic fever. There is no evidence of renal disease.

Inspection. The chest is nearly symmetrical; the left nipple slightly highest; there is no marked arterial impulse or venous distension. The heart's impulse is observed at, and two inches to the left of the left nipple; it is slight *in epigastrio;* respiration is tranquil during a state of repose.

Palpation. The cardiac impulse is considerably increased (systolic and diastolic) and heaving; there is fine diastolic *fremitus;* the apex of the heart is felt two inches to the left of the left nipple. The pulse is of the same character in both wrists, exceedingly irregular and unequal, a few slow, followed by very rapid beats.

Percussion. The area of cardiac impulse is increased both to the left and the right side, corresponding to the visible and felt impulse.

Auscultation. The impulse is slightly heaving; the first sound, of crumpling and rasping character at the base (right and left), the apex, and round the axilla; it is less marked in carotids. The second sound repeats itself four times distinctly. This repetition sound is audible at the right and left base, at the aortic and pulmonic cartilages; indeed, all over the anterior surface of the chest, diminishing in the carotids. It extends also from the apex to the left axilla, and is gradually lost posteriorly. This phenomenon has persisted for some time.

With regard, then, to the repetition sound of the heart, I would remark:—

1. It is a phenomenon subject to variation; in one class of cases at times distinct, at others apparently shading into some modification of murmur.

2. It may be either transient or persistent in character.

3. It is very frequently connected with pulmonary irritation.

4. It generally ceases to be audible while the heart's action is accelerated, to become again distinct as the heart resumes its average rate of speed.

Skoda is doubtless correct in assuming that, in some cases, the divided beat is an element of a murmur; but there are certainly numerous cases in which the reduplication at base or apex is so distinct and unmixed with any quality of murmur, that some more comprehensive explanation of its mechanism has yet to be given.

PRACTICAL OBSERVATIONS UPON THE DISEASES OF THE RIGHT ORIFICES OF THE HEART.*

It is the object of this paper to aid in illustrating, practically, the nature of acquired and isolated obstructive and regurgitant affections of the right orifices of the heart as occurring in the adult subject. The extraordinary rarity with which opportunities present themselves for forming an accurate clinical, as well as pathological history of these affections, renders every complete and carefully recorded case of the very greatest importance to a department of physical diagnosis, in which, simply theoretical views have had, for the most part, to suffice for our guidance. Indeed, in one of our latest and perhaps best monographs upon the diseases of the heart, Professor Bamberger, after asserting that the diagnosis of obstructive diseases of the semilunar valves of the pulmonary artery is based upon the existence of a prolonged systolic murmur having its maximum of intensity over the anatomical site of these valves and extending, most strongly, towards the left clavicle and over the right ventricle, proceeds to state, that the murmur is heard less strongly, or not at all, at the apex, over the ascending aorta, or along the course of the carotid arteries. This definition of pulmonic obstructive murmur is not only theoretical but too exclusive as we shall find. Murmurs are not, so to speak, walled in by their originating arterial tube, but extend, *cœteris paribus,* in all directions, in proportion to their intensity. Exception made of cases in which contraction of the conus arteriosus coexisted, there are very few accurately noted records of isolated and acquired diseases of the pulmonic semilunar valves. The cases of the English observers Bond, Lloyd, Speer, Gordon, Begbie, etc., and those collated by Whitley, are either known or may be readily referred to. Those of continental pathologists being less accessible, I shall take leave briefly to detail. In Frerich's case, one hardly free from tricuspid disease, the subject was a hump-backed and consumptive man, thirty-four years of age. The valvular lesion was the result of endocarditis, and combined the phenomena both of obstructive and regurgitant disease. There was perfect fusion of the valves, forming a rigid, cretaceous, dome-shaped diaphragm, the concavity looking downwards, with a central aperture capable of admitting an ordinary sized goose-quill. The right ventricle was greatly dilated and hypertrophied, forming the apex of the heart. The right auricle was also dilated. The leading physical phenomena were, moderate increase of percussion dulness, feeble, often imperceptible cardiac impulse, intense *diastolic* murmur, having its maximum over the second left intercostal space, extending up the arterial trunks to the neck. The systolic sound* was loud and valvular. The (morbid ?) auscultatory phenomena were limited to the dominant insufficiency: the obstructive disease, though considerable, yielding no systolic murmur. The general symptoms were simply those of the consumptive disease. In Benedikt's case, a woman, aged sixty, was attacked with endocarditis, the manifest result of rheumatic fever. On *post-mortem* examination, fusion of the pulmonic semilunar valves, leaving a central orifice, was found, combined with atheromatous deposit in the pulmonary artery, causing both obstructive and regurgitant disease. The physical signs were, intense systolic *fremitus*, with intense and prolonged systolic and diastolic murmur over the site of the pulmonary artery, and stronger over the right than the left ventricle, but not extending up the neck. The general symptoms were, catarrh, dyspnœa, at times amounting to orthopnœa, pallor, blue lips, ascites and anasarca.

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* Read before the Medical Society of London, April 28, 1862.

While, then, there are a very few recorded cases of obstructive and regurgitant diseases, limited to these valves, I do not know that scarcely a single case of acquired (?) and isolated regurgitant affection of the semilunar valves of the pulmonary artery, *diagnosed during life, and having such diagnosis verified after death,* has ever yet been placed on record.

In grouping affections of these valves, generally, I imagine, that a triple division is the most practical, as it certainly is the most natural. The first division would include those cases in which, the representative sign—murmur—is caused by some body, often of tuberculous nature, situate external to the vessel, and diminishing its area by a pressure, fugitive or permanent.* The second division numbers those cases where either congenital malformation, or the change consequent upon fœtal endocarditis persists, and which, as a rule, though unmistakably impressing the system almost from birth, has, nevertheless, permitted adult age to be attained.†

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* No doubt, aortic.

Lastly, come those rare instances we are to consider, of disease, isolated in its nature, and late in its advent.

The first case in this latter category, is one of obstructive and regurgitant disease. It is, perhaps, rendered more interesting from the fact that, about three years ago it was submitted by me, as a typical case, to the examination of some skilled auscultators, but I think the general disposition was to regard it as one of aortic disease.

Joseph Bright, aged seventeen, slightly built, but of ordinary stature, by trade a mason, came under my care in October 1858. His father and mother are delicate; the latter has disease of the heart, consequent on rheumatic fever. Two sisters and one brother survive, and are strong and healthy. One or two others died in infancy.

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* See paper by the author, *British Medical Journal*, 1856.

† See paper by the author, Op. cit., 1857.

Previous history.—He always enjoyed good health, and was capable of great exertion, until five years ago, when he had a severe attack of rheumatic fever, which lasted nearly six months. Since this attack, he has never felt quite well, suffering from considerable dyspnœa on exertion and slight cough.

Four years ago he became suddenly giddy and fell down insensible in the street. He was picked up and carried home. This seizure lasted, it is said, five minutes. Five months ago he was attacked with hæmoptysis and brought up, he states, three table-spoonfuls of red, clotted blood. The bleeding continued in small quantities. Since this time, he has had almost constant diarrhœa. Two months ago, another attack of hæmoptysis occurred to the amount of about two table-spoonfuls, and the same night, in bed, he had a rather severe epileptic fit. He is also subject to severe epistaxis from very slight causes. There is, moreover, great sensitiveness to the action of cold.

Present condition.—He looks tolerably healthy, the face, at times, perhaps, is slightly dusky and swollen, but the tint is, generally, paler than normal. His fingers present no marked clubbing, but the nails are rather broad and convex. He suffers from dyspnœa on exertion and has a slight, and rather loose cough. His bowels are relaxed, acting three times daily.

Physical examination of Chest.—Inspection. Chest not quite symmetrical. Left nipple, slightly, the lowest; slight fulness of left precordial region. Depression of lower ribs and costal cartilages on either side; very slight and limited visible cardiac impulse, one inch below left nipple, vertically. No epigastric pulsation. No cervical or other venous distension. No abnormal pulsation either in carotids or at episternal notch.

Posteriorly.—The spine is straight. The left base slightly smaller and depressed. Respiration tranquil, superior and inferior costal. The lips are florid.

Palpation.—Slight cardiac impulsive, systolic and diastolic from apex to *epigastrium.* No *fremitus* at apex. Systolic fremitus, silvery but well marked, with its maximum between the second and third left costal cartilages near the sternal border. This thrill is transmitted, diminishing, across the sternum; it is felt slightly at episternal notch, but is lost in the carotids.

Pulse slightly strongest in right radial artery, soft and regular, and always ranging from 98 to 100. The latter point of much importance in a diagnostic point of view.

Percussion.—Resonance every where good, excepting from the left nipple across the sternum; here, the sound is more than usually dull, and the sense of tactile resistance very marked. The area of dulness is about two inches vertically, by three inches longitudinally. The liver and spleen are apparently normal to percussion.

Auscultation.—Intense superficial, prolonged hollow and soft, systolic murmur with its maximum between the second and third left costal cartilages, occupying all the periods, and annihilating the second sound of the heart. This murmur is transmitted with intensity over the right ventricle, still masking the second sound. The murmur decidedly diminishes towards the left apex and, now, unmasks the second sound, which is heard with tolerable distinctness. The murmur is also heard at the superior part of the chest, but louder under the left as compared with the right clavicle. It is very distinct too, at the episternal notch, as, also, *all along the course of the carotids.* It is quite audible over the entire posterior portion of the chest.

These notes were most carefully taken when the patient was first seen, and, on numerous occasions, as carefully re-tested. Since the date named, he was, until his death, constantly under my care. No change whatever occurred with respect to the physical signs, save an occasional variation in intensity. The face and lips, particularly, were unusually dark at times. With regard to the symptoms, the hæmoptysis frequently recurred, and the epileptiform seizures became much more frequent, sometimes of convulsive, at others of syncopal type. The cough and diarrhœa persisted. Well marked symptoms of phthisis gradually supervened, causing death on the 15th September, 1861. The body was examined about twenty hours after death. There was considerable adhesion of both pleuræ. Nearly two-thirds of the right lung were destroyed by vomicæ. Considerable excavation, also, existed in the left apex. Some circumscribed consolidation existed in patches over both lungs, probably the result of pulmonary apoplexy.

The heart is nearly twice the natural size, its tissue very dense and congested. The weight thirteen ounces and a quarter. The vertical diameter of the right ventricle is four inches and a half; the transverse about three inches and a half. The apex is entirely formed by the right ventricle. Near the upper third of the outer surface of the right ventricle, is a patch of old false membrane, about the size of a half-crown piece. Lower down, are two smaller milk spots. The right auricle is in a state of very great dilated hypertrophy, the walls being three times thicker than the left. It contains a very dense, fibrinous coagulum, extending to the end of the appendix, which is, also, dilated and thickened. The *foramen ovale,* partially closed by valve, is very largely patulous, admitting the end of the little finger. The tricuspid orifice is small as compared with the left. The valves are healthy. The walls of the right ventricle are nearly one inch and a quarter thick, and so great is the amount of concentric hypertrophy, that the cavity would scarcely contain a large almond. The ventricular septum is perfect, and three-quarters of an inch in thickness.

The pulmonary artery is nearly double the size of the aorta. Its walls are extremely thin, giving way on very gentle traction. The vessel contained a dense coagulum. There exists the most perfect fusion of the semilunar valves, although the white lines marking the original separation are unusually visible. The valvular diaphragm, thus formed, is funnel-shaped, the concavity looking downwards. The central aperture is round, fringed to the extent of two-thirds of its circumference with minute vegetations, and would admit an ordinary goose quill. The ductus arteriosus is completely closed. The left auricle is nearly normal. The ventricle hypertrophous, its walls three-quarters of an inch in thickness. The mitral orifice and valve healthy. Aorta unusually small.

This case presents some residual phenomena of interest in the pathology of the heart.

It is, indeed, the type of a class, so far as regards the mere morbid anatomy, in which fusion of the valves forms an almost perfect membrane, with a narrow, central perforation. No sudden effort of the ventricle could enable it to deliver its contents during a physiological period of the heart's beat, through the spout of the funnel-shaped orifice ; so that assuming the contraction of the right ventricle (in this case, a gradual squeezing of the blood through a coarcted orifice) and the prolonged murmur, to be coincident in point of time, it is manifest, that the left heart would beat, not only out of proportion to the right, but also as regards the normal mean of ordinary healthy pulsation. This latter result is, doubtless, a compensating effort to amend in point of speed that which failed in regard to quantity and quality of blood; for we cannot conceive that, with the normal ingress of the blood to the pulmonary artery impeded by a curtained wall, a sufficient quantity would be, uniformly, passed into the left auricle. The thickened walls of the left chamber and the undeveloped aorta are, probably, thus, in part, accounted for. If any question should arise as to the non-existence of reduplication of the second sound, consequent on this want of synchronism of the ventricles, it will be found, considering the condition of the valves, that the physical conditions for such phenomenon were simply impossible.

The second sound, heard at the apex, was, clearly, that of the aortic semilunar valves. No murmur, usurping the place of the normal second sound at the pulmonic outlet, existed, in consequence of the absence of any quick and normal contraction, in the thinned and dilated pulmonary artery. The blood, so gradually forced into it, fell back again through so narrow an outlet and in so small a quantity, almost by the mere force of gravity, as to be, practically, without the power of engendering murmur.

There remains, finally, for consideration, the further influence of the immense concentric hypertrophy of the right ventricle. So small was the cavity left, that, even with the aggregate of the influent and refluent streams, it was hardly capable of containing one-third of the average quantity of blood. The narrowing of the tricuspid orifice is not to be considered, I think, as the result of morbid change, but, rather, as the consequence of the gradual increase of the concentric hypertrophy of the right ventricle. In any event, the distension of the systemic and pulmonary veins was a necessary sequence. No case could fulfil, in more exaggerated degree, the theoretic requirements for the production of cyanotic tint. But this was by no means marked. It might, however, have been averted as well as permanent congestion of either liver, or spleen, etc., by the hæmorrhage and other draining, to which the patient was so liable.

It is quite possible that this case might be regarded, by some, as one of congenital, with more recent superadded disease. Such a view it would be well to anticipate. The arguments to be urged in its support, would, I apprehend, be the following:—The perfectly smooth fusion of the valves; the largely patulous *foramen ovale;* and the additional fact, that, in some rare cases, cyanotic phenomena have not appeared until comparatively a late period, or until subsequent disease had added its *quotum* to the pre-existent state. Fusion of valves is, assuredly, not restricted to intra-uterine endocarditis. It may occur in acquired aortic, and there are already grounds for believing in acquired pulmonic disease also. With regard to patency of the *foramen ovale,* it must be remembered that, in subjects dead of the most varied forms of disease, the edges of this canal are often found in a state of imperfect union, sometimes widely patulous, so that patency is quite reconcilable with a state of acquired obstruction of the pulmonic orifice. Here, it probably exercises a compensating influence. Perhaps, too, fortunately for my argument, in the case narrated, the ventricular septum was entire and the arterial canal closed. With respect to the late manifestation of cyanotic *signs* in congenital disease, although this holds certainly true at times, yet the peculiar *symptoms*, more or less marked, of obstructed circulation, are, for the most part, coetaneous with extra-uterine life.

What, again, would constitute the proof of engrafted on congenital disease? The injured valves could scarcely be regarded as a *criterion*, for they exhibit none of the characteristics of disease acting at distant and successive periods. One attack is fully equal to the explanation of the observed results. It may be, doubtless, very difficult to conceive of an attack of rheumatic fever implicating the normal heart, and, so far as regards its orifices, exhausting its entire force upon that of the pulmonary artery. But around the other view, equal, if not greater difficulties crowd. For, while freely admitting that an unsound valve is more obnoxious to injury from a poison having some mysterious elective affinity for its structural elements, yet it is in the highest degree improbable that such a superadded blood poison should have limited its action to the right orifice and have left those of the left side intact, the seats *par excellence* of its ordinary specific action. Keeping, then, fully in mind, the particulars of the case, that a lad, previously healthy, and capable of fulfilling not only the ordinary, but even the laborious duties of life, those of a bricklayer and mason's assistant, gets an attack of rheumatic fever. From this date, the entire cohort of morbid phenomena indicative of obstruction of the orifice of the pulmonary artery develop themselves in all their intensity, and, after death, this orifice being the only one of those of the heart affected, the presumption is greatly in favour of the case being one of acquired disease.

Case ii. This case, though with less of detail, is yet of far greater intrinsic importance, being one of isolated change of the pulmonic valves and artery, causing permanent regurgitant murmur. The following is its history. About three months ago, a young man, aged twenty-five, was admitted under my care at the Royal Free Hospital, affected with disease of the chest. He stated that, four or five years since, he suffered from an illness attended with great pains in his joints. Since this attack, he has suffered from cough, dyspnœa, and, at times, from severe palpitation of the heart. Latterly, being very destitute, and exposed to much hardship, these symptoms have much increased, and he had lost both flesh and strength, and suffered much from night perspiration. On examination, extensive phthisical disease of the left lung was detected. But more than this (in addition to slight impulse, probably given by the pulmonary artery left free by the contracting lung) between the second and third left costo-sternal cartilages, a superficial and well marked, though limited, diastolic murmur was audible, unattended by fremitus, and gradually lessening as the stethoscope was moved from its *maximum* point. The first sound was apparently normal. No collapse of vessels was visible, and the pulse had the ordinary weak and rapid beat of phthisis. Indeed, the general symptoms were for the most part those of this disease.

I pointed out to Mr. Hill, that the murmur originated at the pulmonic semilunar valves. But, I hardly dared trust the diagnosis, so improbable it seemed. After the patient's death, which occurred towards the latter end of January, the diagnosis proved correct. The left lung was condensed by tuberculous deposit, and contained a considerable cavity at the apex. The right lung was in a less advanced state of disease. It was moreover found, that in addition to very extensive dilatation of the mouth and trunk of the pulmonary artery, the semilunar valves were imperfect. I use this term purposely to challenge the fullest discussion; since it might be argued that the condition of the valves to be described was one of original imperfection. For it is shown by pathological anatomy, that where supernumerary valves exist, they are very frequently found in the pulmonary artery. But I think that this specimen, when viewed in connection with the clinical and pathological history of the case, may prove that the division is caused by adhesion occurring at the time of the probable attack of rheumatic fever. But I do not insist upon this point. In this case, the heart is remarkably small, probably, originally so, as there are no signs of atrophy. It rather resembles the heart of a boy of twelve or fourteen years of age, than that of an adult of twenty-five. Its magnitude is scarcely one-third of that described in the preceding case. The weight, after being preserved in methylated spirit, is barely five ounces. The apex is rather formed by the right ventricle. The right auricle and its appendix are much dilated. The walls thicker than natural, somewhat exceeding the left in this particular. Its capacity is about triple that of the left. It contained a large, dense, yellow, fibrinous coagulum, which extended into the appendix. It was most firmly adherent, intertwining among the *musculi pectinati*, and tearing on a firm attempt to remove it. The *foramen ovale* is patent. The orifice valvular, and capable of admitting a crow's quill. The right ventricle is relatively hypertrophied, but not to the extent of the left. The tricuspid orifice, for so small a heart, is much dilated, readily permitting two fingers to pass. The valve is probably incompetent. The left auricle is nearly normal.

The left ventricle, bearing in mind the size of the heart, is in a state of marked dilated hypertrophy. The walls measure five-eighths of an inch in thickness;

the cavity would hold about a small hen's egg. The mitral valve is normal. The aorta is about seven-tenths of an inch in diameter; a tolerably fair proportion to the size of the heart. Its valves are perfectly healthy. When, after removing the heart and vessels, Mr. Hill poured water into the aorta, the column was perfectly supported; but on pouring it into the pulmonary artery the fluid immediately passed the valves, and escaped through an opening made in the right ventricle. It is to the condition of the pulmonary artery, and its valves, that I have to advert. The artery is very greatly dilated; indeed, so much so, that it might without impropriety be described as being in the condition of true aneurism. And it is very probable that the pulmonic orifice is doubly insufficient, primarily, from the imperfection of the semilunar valves, and secondarily, from the dilatation of the arterial mouth. There are no signs of atheromatous change in the vessel, nor is the elasticity of its walls, apparently, impaired. With respect to the valves, their general arrangement and position are normal, but one of the valves is divided, by a short cord, into two unequal portions, and the edges of the adjacent segments are adherent for some distance. They are by this change obviously incapable of normal closure.

In this case, when we consider the influence of one retrograde current from the pulmonary artery, and admit the probability of another through the tricuspid orifice, we, at once, obtain an insight into the mechanism of the extreme dilatation of the right auricle and the hypertrophy of the right ventricle; but it is not so easy to explain the cause of the dilated hypertrophy of the left ventricle. I would simply offer a conjecture, considering the fact as almost certain that the distended right auricle forced some venous blood through the foramen ovale into the left auricle, whether the stimulating effect of this blood might have some influence in causing the over nutrition of the left chamber. Lastly, both these cases are additionally interesting as strengthening the belief that the rheumatic poison may, though with very unequal frequency, affect the right side of the heart even to the perfect exclusion of the left. They both, too, occurred in subjects nearly of the same age, and both terminated in phthisis after a lapse of about four years. This is a different result to what is ordinarily taught. But it will, I imagine, be found on more enlarged experience that imperfection of the valvular semilunar orifices gives a special disposition to phthisis. These cases, certainly, by no means support the well-known views of Rokitansky. It is also worthy of mention that with the greatest obstruction and the most preternatural freedom of orifice, the pulmonary artery was, alike, unusually dilated.

CASE OF A PERSISTENT MURMUR IN THE PULMONARY ARTERY.*

The physical signs of abnormal conditions of the pulmonary artery, as laid down in our text-books, are avowedly those rather of a speculative than of a clinical character. Until within a very recent period, we vainly sought in the pages of our literature for the auscultatory phenomena of cases accurately observed during life. This has resulted rather from the comparative rarity of the cases than any inherent difficulty in their physical investigation. It is not, however, unreasonable to expect, now that the differential diagnosis of the valvular diseases of the heart is so much simplified, that the occurrence of murmur in the pulmonary artery may not eventually prove so unusually rare as was at one time imagined; since it is certain that very many cases of disease of this vessel, revealed by *post mortem* examination, are found in the writings of the most approved writers, both of an earlier and more recent date.

In addition to the valuable communications of Dr. Chevers, we learn that, in an analysis of 100 cases by the late Dr. Clendinning, valvular disease was found sixteen times more frequent upon the left than the right side of the heart. Bizot found, in 157 cases, 9 of disease of the pulmonary artery. Blakiston noted disease of the valves of the pulmonary artery five times in 156 cases. Peacock, in 41 cases of defective development of the valves of the right and left ostia, found 32 occurring in the aorta, and 9 in the pulmonary artery. Swett, in an analysis of 48 cases, found 3 cases of disease of the pulmonary valves. Lastly, Bamberger (Virchow's *Archiv*, 9tes Band, 4tes Heft), in 52 autopsies, found 2 of simple disease of the valves of the pulmonary artery occurring in youths between the ages of 10 and 20 years. Well marked cases of such disease, diagnosed during life, have now been recorded by Hope, Elliotson, Bouillaud, Ormerod, Hamernjk, Frerichs, Benedikt, Dietl, etc.

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* *Association Medical Journal,* 1856.

The case I now venture to publish at least fulfils all the theoretic requirements for pulmonary arterial murmur. It has been constantly under my own observation and that of several excellent observers, for a period of six months, without the slightest variation with respect to the physical signs; and, as the patient still enjoys an average amount of health, there is no reason to delay the physical history of the case, although the anatomical proof be absent. I may add, moreover, that Dr. Sibson has recently examined this case, and concurs in the description of the physical signs.

Case.—Emma Baker, aged 19 years, of sedentary occupation, was admitted a patient of the City Dispensary, February 4th, 1856. She complains of headache after dinner, pain over the lower sternal region, passing through the chest to the blade bone; disposition to faintness, with occasional complete loss of consciousness. She has suffered from dyspnœa as long as she can remember; frequently recurring catarrh, characterised by cough and very considerable expectoration of thick phlegm. Under mental excitement, the ordinary tint of her face becomes deeper, and her lips turn quite black. The face and eyelids appear puffy in the morning; swelling is also observed about the calves of the legs. The kidneys act healthily. Menstruation is tolerably regular. The appetite is good; sleep undisturbed. Her parents are both living, and, with her brothers and sisters, healthy. She has never had rheumatic fever, or any inflammation within the chest.

Inspection.—Her face is round and full, and of dusky tint; the lips are blue, conveying the expression of "facies cardiaca" in an early stage. There is no abnormal pulsation of arteries, or cervical venous distension. The mammæ are large. The finger nails are blue, but the ends of the fingers are natural with respect to shape and temperature. The feet are swollen, and covered with large varicose veins the size of the little finger. The chest and spine are perfectly normal in shape. General nutrition is perfect. She is of the average height and strength. Respirations, twenty per minute.

Palpation.—Slight but well marked systolic *frémissement,* from the second to the fourth costal cartilages of the left side, occupying an area vertically of two and half, and transversely of three inches, is perceived. The maximum site of *frémissement* is between the second and third left cartilages; it extends to that portion of the sternum corresponding to the cartilages named, and is then lost. It also ceases to be felt to the left, after occupying the area named. Not the slightest *frémissement* is appreciable to the right of the sternum, nor even upon the left side below the site indicated. The apex of the heart is not exactly *in situ,* rather inclining to the left of the nipple line, between the fifth and sixth ribs. The impulse is perfectly normal. Not the slightest *frémissement* exists here, or in the carotids. In the episternal notch, slight pulsation is felt. There is no epigastric impulse. The pulse is quite soft, regular, and equal in both wrists, but permanently above the average frequency, generally ninety-six per minute. Not the slightest pulsation exists over the area of thrill.

Percussion.—The percussion sound is uniformly clear over the lungs, as also over the whole sternal region. No enlargement either of the right or left cavities of the heart is indicated by percussion; but the result may be modified by the large size of the mammæ.

Ascultation.—Between the second and third left costal cartilages, the normal first and second sounds are entirely replaced by one extremely superficial, soft, though very loud and almost continuous systolic *bruit*, gradually attaining its maximum of intensity, and then gradually decreasing, exactly imitated by the *bruit de diable.* There is a slight intermission at what should be the end of the long interval of silence, or the murmur would be absolutely continuous. The maximum intensity of the murmur is at the lower border of the second left costal cartilage, one inch distant from the left border of the sternum. The murmur is comparatively distant and slighter during deep inspiration, but becomes again exceedingly loud and blowing into the ear during forced expiration. It is transmitted, though with greatly diminished intensity, to the root of the pulmonary artery and body of the right ventricle. In the epigastrium, both normal sounds are audible, though still mingled with distant murmur. At the left anterior surface, all *bruit* ceases, almost abruptly, below the fourth cartilage; and now the second sound begins to be audible. At the left apex, both sounds are perfectly normal, with reference alike to rhythm and timbre. Not the faintest murmur is heard here. The impulse, also, is perfectly natural. Upon the right side of the chest, both sounds are distinctly heard, the first being accompanied by distant *bruit;* the second very distinct and clear, which character extends nearly to the left edge of the sternum. At the episternal notch, and over the carotids, the second sound is also particularly distinct, and the first sound unmixed with murmur. Posteriorly, about the superior border of the scapulæ, distant *bruit* appears audible at intervals, especially during deep expiration. Over the remaining portion of the chest, faint though normal *tic-tac* can be heard. Both *frémissement* and murmur become considerably intensified upon movement. No jugular venous murmur is audible. The respiratory murmur is tolerably pure, but apparently louder over the left side.

From the period of her admission to the present date, the physical signs have undergone no change. Her constitutional condition has undergone some modification. She has exhibited considerable variation with respect to the semi-cyanotic tint; has once had hæmoptysis, and, some months subsequently, considerable anasarca of the lower extremities.

Remarks.—The weight of proof resting upon me, it may perhaps fairly be required that I should state the reasons why this case should be regarded as an affection of the pulmonary rather than of the systemic artery, since an error in diagnosis might so readily be made, more especially when it is remembered that Hope, and, more recently, Bamberger, have related cases of aneurism of the concavity of the arch of the aorta, which, by pressing upon the pulmonary artery, engendered a loud and harsh systolic murmur immediately over the anatomical site of this vessel. The undermentioned points would appear to me to constitute the principal arguments against the existence of aortic aneurism or other disease.

1. The general history of the case, which testifies to the existence of more or less dyspnœa, lividity of lips, etc., as long as the patient can remember.

2. The age and sex of the patient. Aortic aneurism is comparatively rare in women. Of 189 cases analysed by Bizot, 171 occurred in males, and only 18 in females. The mean age, also, of aortic aneurism, is, according to the statistics of the best observers, between 30 and 40. This patient is under 20 years of age.

3. The entire apparent integrity of the aortic system, as evidenced by percussion and auscultation, and the absence of any well marked collateral sign characteristic of aortic disease.

4. The absence of the ordinary causes of disease of the heart at this period of life, rheumatic fever, thoracic inflammation, etc.

5. The difficulty of conceiving any other extrinsic cause of displacement of the aorta, without attendant physical signs. And even the rare occurrence of transposition of vessels would fail to explain a systolic murmur. Skoda has stated that, in some cases of mitral disease, a murmur is heard over the pulmonary artery. The same proposition may be enunciated with respect to disease of the aortic valves. Nothing is more common in insufficiency of these structures than for murmur to be heard over the pulmonic cartilage, covering more or less completely the normal sound of closure*

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* I believe this to be invariably true with regard to musical diastolic murmur, and most frequently so with regard to ordinary *souffle*, when at all intense.

—a clinical fact which finds a ready explanation in the circumstance that, from superposition, vibrations are readily transmitted from a defective aortic orifice along the course of the pulmonary artery; and it is matter of astonishment to me that authors have affirmed the differential diagnosis of diseased aortic valves, on the more than doubtful premiss, that the clear sound of closure of the pulmonic valves is still audible at the pulmonic cartilage.

Assuming, then, the evidence adduced sufficient to negative the hypothesis of aortic aneurism, etc., and the murmur to be actually generated at the pulmonic artery, the question yet remains open, whether the murmur be caused by some alteration of the surrounding tissues, or some inherent condition of the vessel itself; that is, whether the proximate cause be of an extrinsic or intrinsic nature. On the first supposition, could tuberculous infiltration at the anterior edge of the lung, or cartilaginous growth within the pericardium, as observed by Elliotson, engender murmur by pressure upon the trunk of the artery? If tubercle, an isolated mass might have been formed in the precise situation, and, subsequently, by the venosity of the blood induced by pressure upon the artery, the further progress of tuberculosis prevented in conformity with Rokitansky's theory of antagonism.

Is there a probability of its being caused by aneurism of the pulmonary artery? In deciding upon this question, we cannot, as in the case of aortic disease, adduce the negative testimony of age and sex; since, according to Walshe, aneurism of the pulmonary artery has been observed in girls under twenty years of age. Indeed, Blakiston records a most instructive case in point. Against this view may be stated—

1. The absence of any appreciable disease of the aorta, with which, according to Rokitansky, aneurism of the pulmonary artery is almost invariably associated.

2. The absence of pulsation and percussion dulness.

3. The general history and progress of the case.

Finally, may the murmur owe its origin to some congenital cause—patency of the ductus arteriosus, open *foramen ovale,* with imperfection of the *ostium* of the pulmonary artery? In a very large number of cases analysed by Gintrac, Louis, and Bouillaud, this double imperfection was found to exist: indeed, so constant was the sequence, that Louis, in reviewing the varieties of structure of stenoses of the pulmonary artery, arrives at this conclusion:—'Il semblait donc résulter de tout ce qui précède que le trou de Botal, à une époque quelconque de la vie, la perforation de la cloison des ventricules et le rétrécissement de l'artère pulmonaire, doivent être considérés comme des dispositions congenitales.'

I believe, upon consideration, that this latter mode of origin, or some modification of it, will prove the correct one. This opinion is supported by the *com-*

memoratifs of the case; the more or less habitual dyspnœa; the recurring bronchial irritation; the semi-cyanotic tint of face and nails, varying in intensity from time to time; the changeless character of the physical signs, and, with slight exception, of the constitutional phenomena.

One other point in this case is deserving of comment, viz., the apparent absence of any marked structural change of the right chambers of the heart. The absence of such change may possibly be explained upon the assumption of a patent condition of the ductus arteriosus. Upon reference to Gintrac (*Observations et Recherches sur la Cyanose,* 1824), it will be found that, in cases 4, 9, 13, 19, 22, 25, the duct was open; and no mention is made of the enlargement indicated, which is specially pointed out in some of the series.

ADDITIONAL OBSERVATIONS ON TRANSPOSITIONS OF THE GREAT VESSELS OF THE HEART.*

In the current volume of the *Medico-Chirurgical Transactions,* a case of transposition of the great vessels of the heart is detailed by me, which was associated with simple patency of the foramen ovale. This case is, moreover, compared with the others on record, three in number.

In these four cases, were it not that some interchange of blood must have taken place through the foramen ovale, two circulating systems, perfectly distinct, would have existed. The right, or aortic system, would have constantly circulated black blood through all the organs connected with it, the muscular tissue of the heart inclusive; and the left or pulmonic system would have as constantly circulated oxygenated blood through the lungs. Indeed, even with a patent foramen ovale, the actual state of the circulation must have been nearly as described, so far as regards the systemic vessels. But, as Meyer† has pointed out, there are mechanical causes for an easy transit of blood from the right to the left auricle. The blood from the left or pulmonic heart, while circulating through the lungs, receiving no substantial additions, would have exhibited a constant tendency to concentration from loss of its watery portion from pulmonary exhalation. The absolute quantity, consequently, transmitted to the left auricle would have been a constantly decreasing one, and inadequate to the physiological distension of the chamber. On the other hand, the blood of the caval system, constantly receiving substantial addition from its tributaries, would exhibit as constant a tendency to repletion of the right auricle. Hence the necessary result would be, that any surcharge of venous blood would enter the left auricle through the patulous foramen ovale to restore the equilibrium. That this result did follow in the author's case is probable from the increased thickness of the walls of the right auricle.* It would, he imagines, be greatly more difficult to explain the manner in which oxygenated blood could pass from the left into the right auricle.† But that such passage was effected, we must admit.

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* From the *British Medical Journal,* 1863.

† Virchow, *Archiv,* B. 12, 4tes-5tes Heft, p. 371.

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* Upon this point, Cruveilhier, *Anat. Path.*, t. ii, pp. 477-84, makes the following statement. In hearts presenting no other complication than simple, though ample communication between the auricles, the following changes are observed, general enlargement of the right cavities of the heart and dilatation of the pulmonary artery and its branches. The left cavities stand in striking antithesis to those of the right side, being below the average size.

† For arguments upon this matter, consult particularly Louis, *Research. Anat. Path.*, p. 337; and Allan Burns, *Observations on Diseases of the Heart*, p. 15.

Such also seems to have been the opinion of most of the observers of the recorded cases. After carefully considering the whole question, the author is unable to give a satisfactory answer to the following question: How is the absence of cyanosis during the first three months, and its comparatively sudden supervention at the close of that period to be explained ?*

There are no *data* from which to infer the actual period of closure of the arterial duct.† Even if there were, they could hardly serve to explain the phenomena in question. The small quantity of blood transmitted through this channel into the aorta during three months would scarcely have tinged the mass of venous blood in the systemic vessels. Its closure, consequently, at this period, could hardly in any way have influenced the production of cyanotic tint.‡

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* For some interesting examples of malformation showing the length of time prior to the supervention of cyanosis, see Naumann, *Klinik*, B. 2, s. 231 *et seq.*; also Meckel, *Path. Anat.*, B. 1, s. 441; and Louis, *op. cit.* p. 340.

† For the most detailed accounts of the period of closure, see Haller, *Element. Physiol.* t. viii, p. 10; and Billard, *Mal. des Enfans*, p. 486-94.

‡ We must bear in mind that, no obstruction of the orifices existing, the necessary permanence of this channel was not essential. The cases of the series show that one permanent fœtal way suffices. But, even if thought necessary to reserve this point, the explanation of cyanosis is, certainly, not afforded by the more ordinary theory of pulmonary stasis. The theorem of Cruveilhier (*op. cit*, t. iv, p. 374) would demand modification. The tint must obviously be due to the circulation, through the systemic vessels, of non-oxygenated blood. For the consideration of the question how far the blood, *per se*, can influence cyanotic tint, consult

In the case recorded by Gamage, the child was born perfectly healthy. No cyanosis supervened for some days; yet the arterial duct seems *never* to have existed. The foramen ovale was largely patent. In these cases, one is almost led to think that perhaps one or more organs, probably with varying activity, have for a time performed some work of compensation—the liver, kidney, skin,* mucous tracts,† or thymus gland. In very many cases of cyanosis,‡ this latter organ remains unusually large.§ According to the very careful experiments of Friedleben, related in his work upon the *Physiology of the Thymus Gland*, a diminution of carbonic acid naturally exhaled, to the extent of 14 per cent., occurred in animals deprived of this organ. Furthermore, in support of this view of the vicarious action of the thymus gland in malformation of the heart, we have the concurrent testimony of Mekel, Sandifort, Duretus, Cailliot, and Burns. This hypothesis, perhaps, receives some support from the author's case, in which the organ was very large.

Causes of Transposition of the Great Vessels.—There is, as may be well supposed, great uncertainty attending the determination of the proximate cause of trans-

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Kreysig (*opus posth.*), *Krank. des Herz. im allgemein und auf ihrer erst. Entwickelungstufe*, pp. 232-3; also, Hamernjk, quoted by Henle, *Handbuch der Rationel. Pathol.*, B. 2, 17.

* Farre, *Malformations af the Heart.*

† Nasse, *Reil's Archiv.*

‡ Gintrac, *Sur la Cyanose.*

§ It must be admitted, however, that a case by Nasse, *Lichenöffnungen Erst. Theil*, p. 167, opposes this view; he expressly states 'Von der Thymus war nichts zu finden'.

position of the vessels. The very important results that have followed the study of the changes which the vascular arches undergo in certain cases of abnormal development of the branches of the main vessels of the chest, have hardly yet extended to the explanation of the phenomenon of transposition of the main vessels themselves. Kürschner,* so far as I know, is the chief upholder of the view that faulty metamorphosis of the vascular arches may determine transposition of the heart and great vessels. The researches even of Von Baer, Räthke, and Bischoff, in vascular embryology, have not removed the difficulty. Von Baer† has indicated the manner in which the rotation of the embryo in the yolk to the right determines the transposition of the heart to the right side; but he has not shown how the main trunks alone undergo transposition. Perhaps the more commonly received opinion is, that the phenomenon is dependent upon some irregular torsion of the aortic bulb after its division into the systemic and pulmonary trunks. There is one other very interesting question connected with these and other kindred changes, which is, how far they may be influenced by external exciting causes. D'Alton‡ has given good reasons for supposing that unnatural compression of the abdomen during pregnancy may be productive of the change. At the same time, he admits the difficulty of the explanation.

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* *Vide* Friedberg, *op. cit.*, p. 96.

† *Vide* Von Baer, *Erster Theil.*

‡ *Op. cit.*, p. 23.

Prognosis.—Considering the serious nature of transposition of the great vessels of the heart, and, occasionally, the apparently insufficient nature of the compensation afforded—viz., simple patency of the foramen ovale—it is difficult to conceive how life could be long maintained. Its duration, however, appears to range within comparatively wide limits. While in some cases death has happened almost in a few hours, in others the subject has survived even for years. In the case I have narrated, the child lived two years and eight months. In that recorded by King, two years and nine months were attained. These, with one most remarkable exception, are the longest known instances of the duration of life under such circumstances. This exception is in the case recorded by Nasse.* The subject actually reached the age of nineteen years and some days. Here the foramen ovale was patent, but only to the extent of admitting a crow's quill. The ventricular septum was also defective to the extent of nine lines. From the anatomical results in this remarkable case, it might be inferred that deficiency of the ventricular septum was much more favourable to the prolongation of life than might perhaps appear from an analysis of the cases of transposition extant. But, in some cases with both auricular and ventricular septa defective, life has not been long maintained. In the case observed by Meckel of transposition of the vessels occurring in a lamb, the foramen ovale, ventricular septum and arterial duct were all open; yet the animal only lived two days.*

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* *Leichenöffnungen, Erster Theil.*

Diagnosis.—It has been generally considered impossible to arrive at a knowledge of the existence of transposition of the vessels during life, either by the symptoms, or by the usual methods of physical investigation. And, considering the extraordinary rarity of the affection, it is certainly probable that this opinion will long maintain its ground. Still, reflecting carefully on the morbid anatomy of the cases recorded in the third series, there are, unless I greatly mistake, at least the elements existing for a possible diagnosis. Cases of transposition may at once be separated from the more ordinary congenital malformations connected with obstructive change of the orifice of the pulmonary artery. In this common class, the narrowed outlet reveals itself mostly by murmur, and by inference we estimate the coincident or consecutive changes. In transposition of the vessels, abnormal though the circulation be, yet in no sense can it be said to be obstructed. The cyanosis, as before hinted, when existing, can certainly depend upon no pulmonary stasis from impeded egress through the pulmonary artery. The blood freely passes the outlet, and permits the valves to close normally after it. But it is from the manner in which these valves close that some information may possibly be derived. If we glance at the cases of the third series (and at some of the first and second), it will be perceived that, in all the former, the pulmonary artery was absolutely or relatively much enlarged. In D'Alton's case, and in my own, this is obvious. On examining D'Alton's plate, it will be seen that, as in my case, the pulmonary semilunar valves are remarkably large.* These valves, then, theoretically, should close with a sound proportionately louder and more marked than those of the aorta. This occurred practically in the case now brought forward. It would, therefore, appear that if, in a case presenting the ordinary symptoms of malformation of the heart —without pulmonary or other disease to complicate it —the heart, though increased in impulse, has its sounds clear and normal, save in regard to an unusually marked second sound at the second and third left costo-sternal cartilages, we may be justified in suspecting that it is one of transposition of the vessels of the heart.†

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* For much valuable and interesting detail respecting the epochs at which death occurs in cyanosis generally, I must again refer to the elaborate and practical essay of Nasse, in the *Archiv für die Physiologie* of Reil and Autenrieth, Band 10, s. 239 *et seq.*; also Horner, *Diss. de Cyanosi*, 1832.

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* Only one exception, so far as I know, has been found to the cases contained in the fourth series (*i.e.*, with closed duct). This case is described by Dr. Kelly, *Transactions* of the Pathological Society, vol. 22. In another case by Dr. Pye-Smith, *op. cit.*, vol. 23, the pulmonary artery was enormously dilated, its valves of normal appearance but larger than those of the aorta; here, however, the duct was patent.

† The case recorded seems also to bear upon the question of murmur as engendered at the foramen ovale. Here, if ever, the condition would probably have existed for the production of such murmur from the apparent necessity of a current through the foramen, guarded, in part, by the remains of the valve. Murmur, however, was at no period detected.

FURTHER NOTES ON PULSATING TUMOURS OF THE NECK.*

I SUBMIT the details of two additional cases of Dilatation of the Innominate Artery, which, added to those already published by me, may enable some general conclusions to be drawn respecting this affection.

The first case occurred in a patient of Mr. Parrott, of Enfield, and which I saw with him on two occasions during the month of April last. Mr. Parrott informed me that his attention was first directed to the tumour in December 1868, when called to the case in consequence of several severe attacks of epistaxis, in one of which he was compelled to plug the nostril before the hæmorrhage could be arrested. He considered this circumstance noteworthy, as showing, perhaps, a tendency to disease of the vessels. The artery at that time was pulsating very strongly, from the sternal notch to the origin of the carotid. It was not quite clear to his mind, at this period, whether the aorta was implicated, but he considered that it was. There were no accurate means of knowing how long the tumour had existed, but, in his opinion, it probably originated about that time.

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* Read before the Medical Society of London, Nov. 30th, 1869, and published in the *Lancet*, Jan. 1, 1870.

I took the following notes when I saw the case. The patient was a middle-aged widow, somewhat corpulent, and of sallow complexion. A tumour, which had been observed about four months, very large and prominent (but appearing larger than it really was from a large collection of adipose matter in front of it), completely filled the episternal hollow, passing up somewhat in front of the trachea, and from thence obliquely across the right side of the neck. The impulse was very expansive and forcible, but unattended by thrill; the collapse did not feel quite complete, but this might be explained, possibly, from the collection of adipose matter mentioned. The right carotid and right radial pulses (I speak doubtfully) seemed a trifle weaker than those of the opposite side. A double murmur was audible over the tumour, and also down the aorta, to its origin. The heart's impulse was by no means great, nor was its apex lowered. The condition of the pupils could not be noted, as the patient had lost the sight of the right eye for some years.

In a recent communication from Mr. Parrott (Nov. 25th) he states :—" The patient has lately had three attacks of hæmorrhage, always occurring in the night or early in the morning; on awaking, she has blood in her mouth. I do not think the tumour has increased in size since you saw it; she has occasionally some pain in it, but this is always relieved by the application of cold."

I ought, by the way, to mention that this patient was seen in the early stage of the affection by a London surgeon of repute, who suggested the application of instrumental pressure; but the patient either could not or would not persevere in its use.

For the next case I am indebted to the kindness of my colleague, Dr. Rickards. It is a case the more interesting as, in addition to innominate dilatation, there is cirsoid aneurism of the left carotid.

Mrs. G——, admitted under my care at the Royal Free Hospital in July 1869, is a widow fifty years of age. Her complexion is slightly pallid, but otherwise natural. There is some amount of spinal curvature. She has always been of delicate constitution, and had formerly to work hard. The cause of her father's death she does not know. Her mother died of diseased liver. Her two brothers died of disease of the heart, at the respective ages of fifty, and fifty-two. About seven years ago she first felt a beating in the hollow of the throat, and suffered from rheumatism (as she expresses it) of the head and neck. She still suffers much, at times, from this pain, which affects both sides; also from cough in the morning, shortness of breath occasionally, especially on ascending stairs, and from palpitation of the heart on any excitement. On inspection, the left jugular vein is seen distended, but without pulsation. The superficial veins coursing over the left side of the chest are more prominent than those on the corresponding side. The pupils are equal and of natural size. The right radial pulse was stronger than the left when first examined. The swelling of the left carotid artery (the coats of which feel much thickened) involves the upper half of the vessel. Its prominence is very marked about two inches below the ramus of the jaw, as is also the locomotion of the artery, which here advances at each diastole with a rapid and very forcible forward projection alongside the tracheal edge of the left sterno-mastoid muscle, and then as rapidly retreats during the succeeding systole. The tracing of its impulse has been taken by Dr. Hawksley with his stetho-sphygmograph, as also those of both radial pulses. The latter agree in every particular with those taken at the hospital. A pulsating swelling, about the shape and size of a date, is visible, emerging from behind the right sterno-clavicular articulation, and occupying the hollow of the neck. It extends upwards from beneath the inner head of the sterno-mastoid muscle to its outer head, taking a somewhat oblique direction. The impulse is strong, expansive, and liquid during arterial diastole; during systole the swelling perfectly subsides. The heart appears to be displaced horizontally; its apex is situate near the fourth rib, one inch above the left nipple; and there is dilated hypertrophy of the left ventricle. The cardiac impulse is synchronous with that of the tumour and of the radial pulses. No thrill anywhere exists. The right radial pulse, formerly strongest, has appeared of late occasionally to be slightly weaker than the left, but both are perfectly regular, soft, of normal frequency, and without any collapse. The sphygmographic tracing, however, tells plainly of increased arterial resistance. At or just below the right sterno-clavicular junction, a double murmur exists, having here a maximum. The first arterial diastolic murmur is soft and somewhat blowing; the second arterial systolic murmur is a little shorter in duration, and of hoarser character. These murmurs, scarcely changed, are audible in the swelling and right carotid artery, but weaker down the aorta. Immediately under the uppermost part of the sternum a musical murmur of intensity is heard at times, and loudest during arterial diastole; it was of remarkable intensity when Dr. Sibson examined this case with me the other day, and we both consider it of venous origin. About one inch above the left nipple, and corresponding to the site of greatest cardiac impulse, exists a second maximum focus of double murmur. The systolic murmur here is most intense, being at intervals of nearly musical *timbre,* and traceable vertically upwards to the right clavicle, and obliquely downwards nearly to the ensiform cartilage. The second murmur is not so loud, neither is it so widely heard. They diminish somewhat as they are traced midway up the aorta, so that, apparently, a minimum point exists between the two maxima foci. At the left axillary line the murmurs lose all their intensity, and are nearly replaced by normal tic tac, except that the first sound remains faintly murmurish. No murmur is audible at the lower angle of the left scapula. On the left side of the neck a closure sound of the sigmoid valves apparently is heard.

Now, are these murmurs of aortic origin? Such murmurs are occasionally well marked at and near the apex of the heart, and, in exceptional cases, the return murmur may weaken upwards in the aorta; the pulse is perfectly regular, and the absence of collapse might be explained by some coexisting contraction. But we should be perplexed in accounting for the absence of a more defined murmur with the upward current. The sphygmographic tracing wants its characteristic crotchet. But our greatest difficulty, indeed an insurmountable one on the hypothesis of aortic disease—of insufficiency—would consist in accounting for the closure-sound of the semilunar valves in the vessels of the left side of the neck; unless we were to hazard the very speculative conjecture that, from the displacement of the heart, the great vessels might be so compressed as to allow the sound of the pulmonic sigmoids to be conducted so far. Are the murmurs mitral—that is, obstructive and regurgitant? There is an entire absence of the general symptoms of such affection: the impulse-beat of the heart, though forcible, is steady; and the pulse is entirely free from that instability so characteristic of such disease. Can a double lesion coexist, and the conjoined signs modify each other? It is the existence of doubts and difficulties incident to cases like this that must plead my excuse for digressing somewhat from the main subject-matter of my paper to discuss, as briefly as possible, some points connected with the pathology of cardiac murmurs and sounds. It has generally been maintained that comparatively loud murmurs, systolic or diastolic, heard at the apex of the heart, belonged almost exclusively to the affections of the mitral orifice; but I believe that the apex may be at times a common focus of aortic and mitral murmurs. Marey has experimentally demonstrated, what had long been clinically known, that a systolic murmur of intensity may be heard at the apex in constrictive disease of the aortic mouth, propagated by the back current; and I would furthermore add, from personal experience, that when insufficiency occurs in young persons where the aorta is still resilient, a strong diastolic murmur may be heard at and below the apex —indeed, I believe that often the stronger the diastolic murmur in this situation, the greater the reason to suspect aortic insufficiency. That a diastolic murmur occasionally attends constrictive disease of the mitral orifice is well known, and at times with an amount of intensity and thrill which is difficult to explain, remembering that it occurs during a period of cardiac repose, and, consequently, during the passive flow of the blood-current. It appears to be conditioned upon the amount and tension of the blood in the auricle, the tonicity of its walls, and the shape and condition of the obstructing outlet. So that murmur —*qua* murmur—at the apex cannot enable us to achieve precise diagnosis; for, as mitral diastolic murmur may be here strong or weak, so may aortic regurgitant murmur be strong or weak likewise. To effect the desired precision, we must auscult the aorta; observe the amount of collapse of the superficial arteries; seek, when practicable, the intermittent murmur of Duroziez in the femoral artery; study the tracing, and note the regular though special pulse in uncomplicated cases; and also mark the usually unimpaired state of the general health. For in aortic as in moderate mitral insufficiency, if the compensation is simply physiological, and the patient able to observe proper hygienic rules, an average state of health may be maintained for years.

Upon one or two other points connected with the pathology of the mitral valve I would offer a few remarks; for this is the stage upon which the battles of cardiac auscultation have been principally fought of late years, and, indeed, with the one exception of sudden death in insufficiency of the aortic valves, it is here that some of the most fatal incidents of cardiac disease occur. Comparatively recently, much attention has been directed in this country to the murmur known as 'presystolic', and within the last week or two it has been made the subject of a clinical lecture by the able physician of the Charing Cross Hospital. This murmur, however, was pointed out and taught by the highly-accomplished Gendrin when I was a student in Paris some five-and-twenty years ago. It has there been recognised ever since; while his 'peri-systolic' and 'peri-diastolic' murmurs are now almost forgotten terms.

The 'presystolic' murmur is supposed to indicate constrictive disease of the left auriculo-ventricular orifice; and that it does so, when well marked, is manifest. But its value would be greater if the first sound could be heard immediately consequent, to afford the necessary demarcation. But here comes the difficulty in most cases. Although regurgitant disease may exist without contraction, contraction without some regurgitation is, to say the least, a most exceptional occurrence. I can scarcely call to mind such an example. A valve may be rigid, partially calcified, and capable of but little movement—almost fixed at an angle in the ventricle; or the valves may be fused together, forming a funnel-shaped orifice with its apex pointing downwards, and lowering the aperture of communication, as Bouillaud has so well described; or the valves, fused together, may meet at the plane of closure with a rounded aperture barely capable of admitting an ordinary quill; or the chink may be transverse and studded either with calcified spiculæ, or fringed with soft and spongy vegetations, or modified in other ways. But, whatever the form, some amount of regurgitation must, as a rule, occur; for it is not conceivable that a constriction which would admit blood to pass, under the comparatively small power exercised even by a hypertrophous auricle, would not allow some escape back into its cavity during the systolic contraction of the ventricle. Hence, immediately consequent upon the cessation of the abrupt auricular contraction, is heard the loud, harsh, and prolonged murmur of ventricular systole — a murmur, at times, extending through the period of the lesser pause. Again, the ventricular follows the auricular contraction by an almost instantaneous and vermicular action, as if the chambers were one continuous cavity. In a scarcely appreciable fraction of time, the ventricle tightens on its blood, alters its diameters, and assumes a globular shape before it acquires impetus to force the aortic valves. During this preliminary stage, and before the ventricle has acquired the full swing of its power, if a semi-mobile valve, fixed at some angle in the chamber, existed, it is conceivable that a slight murmur or 'susurrus' might be generated, instantaneously followed by the louder and harsher murmur of regurgitation.* Such a murmur would, in a sense, be presystolic, and fuse into the murmur of auricular contraction. At all events, the latter murmur must, when present, be most fugitive; it could not, provided the auricles contract synchronously, exceed the period of their contraction, which Marey has experimentally shown to be so brief; and not fugitive alone, for it is most capricious in its occurrence.

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* From subsequent experience, I am still inclined to believe that this explanation holds good, at least for some cases of so termed pre-systolic murmur. The subject, however, has been most fully and ably treated by Dr. Charlewood Turner in St. Thomas's Hospital Reports, 1876. The date of my paper, however, seems to have escaped his attention.

From anatomical considerations, we infer that, in the physiological condition, both auricles and ventricles *must* alternately contract together: however slow the period of a revolution, or however fast, there is a perfect synergy of the muscular fibres. The chambers, in alternation, fill together and contract together, and a given repletion is probably the condition of their contraction. But, in some pathological conditions, when, for example, the left auriculo-ventricular orifice is converted into a narrow and unyielding chink, it seems highly improbable that both ventricles can fill during the same interval; the blood must here pass through so devious a track in a very diminished and uncertain stream into the left ventricle, and in quantity insufficient to impart the requisite distension stimulous to contraction, even under the compensating aid of an hypertrophous auricle. It is fairly outdone by its less obstructed neighbour, and, being unfilled, is later in its contraction. If this reasoning be consequent, the isochronism of the chambers being perverted, the phenomena of reduplication at the apex would be developed—or scarcely reduplication, for the later sound is a murmur; indeed, the so-called 'reduplication' has been considered by Skoda as a divided rasp sound. If such perverted action of the ventricles really occurred, it might be tested by what must of necessity result, true reduplication of the sigmoid valves at the base; while, on the other hand, if only simple regurgitation occurred, reduplication of the base sounds only would be heard, because the ventricles would both contract together, but the aorta, receiving a diminished stream, would lag behind its more amply distended fellow.

If, on the other hand, this view of a prolonged auricular effort to fill a nearly closed ventricle be rejected, and anatomy enforces its teaching that the chambers not only do, but must, contract in unison, then the attempt at compensation on the part of the auricle can be of little, if of any, avail, for a sudden impulse against a nearly occluded outlet would not only react injuriously upon the impelling agent, but induce an abortive effort, a *faux pas*, on the part of the left ventricle. In simple regurgitation through the mitral orifice, the compensating hypertrophy of the right ventricle, by increasing blood-tension in the pulmonary artery and veins, may lessen the amount of the regurgitating column; but in a case of constricted orifice, such compensation is of little use, if not of positive injury. The ventricle cannot aid the left auricle in disemburdening itself of its large amount of blood, but places the lungs in the most perilous of all positions, having blood forcibly impelled into and through them against a nearly unyielding barrier, and, therefore, inducing the very worst symptoms of lung disorganisation.

I would say, then, with especial reference to these rarer murmurs and sounds, that there are accurate observations and truths embodied in the determination of all their phenomena; but they are not, in my judgment, such truths as we can often make practically available in our ordinary dealings with the diseases of the heart. They constitute the refinements of auscultation. The rarer murmurs and sounds are not only of fugitive, but of changeable character. The presystolic murmur of to-day may become systolic, or even diastolic to-morrow. All murmurs, again, are often obscured by the bronchial râles their causes have originated. In some, also, of the most advanced stages of mitral valve disease, such is the irregularity of the heart's action, and the disturbance of its rhythm, that scarcely tone or murmur can be at all located, much less the precise moment of their production determined; all is fused into a disorderly series of almost indeterminate sounds.

Fortunately, it does not materially influence our practice to know whether the mitral orifice is preternaturally constricted or whether it is preternaturally patent; the backward and successive steps to ill begin in both conditions from the auricle, and the ultimate results are nearly identical; for a contracted orifice stands, to the systemic system, in the relation of an insufficiency, the chief difference being that the left ventricle, at times shrunken from defective supply, cannot afford the compensating aid it does in simple regurgitation.

So that, to my mind, it is infinitely more important to discover, if possible, simply which orifice in the left chamber is really affected, or whether both are involved —more important to measure the extent and direction of the heart's increase, to endeavour to estimate the measure of its contractile power, to ascertain what amount of blood is transmitted to the systemic vessels, and to watch for the gradual transference of vascular tension from the arterial to the venous system.

With respect to the general diagnosis of pulsating tumours of the neck, as I have elsewhere attempted a description, it is only left to remark that cases resembling those now described can be confounded with but few affections of the vascular system proper. Indeed, advanced senile degeneration of the ascending aorta and its arch, and ordinary aneurism of this vessel and its larger branches, are almost alone likely to offer any difficulty as regards diagnosis. Venous varix could hardly be supposed capable of being mistaken for arterial dilatation, did we not find (rarely enough, it must be confessed), that when such affection is of long standing, and the veins are tense, full of firm coagula, and their coats exceedingly thickened, they may receive an impulse from the underlying artery which might, possibly, for a time mislead. Hodgson quotes a case (recorded by Cline) where a large pulsating tumour existed in the neck, and eventually proved fatal by bursting. The sac was formed by the internal jugular vein with the carotid artery, lodged in a groove at its posterior part.

Degeneration of the arterial system is, as we know, one of the common attendants upon old age. The aorta becomes rigid from atheromatous and calcifying change ; it then dilates locally or generally, and often to an extraordinary extent. But the dilatation is secondary, and is caused by the direct impulse-stroke of the enlarged heart those tubes themselves had primarily provoked. The bony arch of the aorta can occasionally be felt on pressing the finger deeply into the episternal notch. There is often marked collapse of the vessels of the neck, and frequently an intensely harsh murmur over the aorta. These local changes, however, are but parts of a more general one ; the visible systemic arteries are locomotive and tortuous, their coats thickened and rigid, rolling freely under the compressing finger ; and Marey has taught us how such change is further revealed in the obtuseness or prolonged level of the summit, and, as a rule, in the total absence of dicrotism of the sphygmographic tracing.

In the cases I have brought forward from time to time, the position and limitation of the external tumour to the innominate artery, its isolation as regards impulse from the greater part of the arch, as shown by the absence of pulsation on pressing down the finger behind the left sterno-clavicular articulation, the absence of *harsh* murmur over the aorta, the absence of collapse until a later period, and the absence of the signs of more general senile change, constitute points of difference. The dilatation of the aorta in these cases also appears to me to be primary; the soft blowing murmur is for a time limited to the upper portion of the aorta, and the sigmoid valves for a time accurately close. Gradually, however, the dilatation extends to the mouth of the aorta, and the valves become simply inadequate to entirely close the enlarged aperture. Double murmur and collapse now occur, and the heart moderately enlarges to effect the necessary compensation.

Lastly, this affection is distinguished from ordinary aneurism, with which it has been at times confounded, by the intermitting character of the tumour—very expansive, tense, and liquid during arterial diastole, with perfect collapse during arterial systole,—this collapse determining the absence of clot; by, as a rule, the unchanged character of the carotid and radial pulses—if any change, the right radial for a time the stronger; also by the absence of influence upon the tumour of pressure upon carotid and subclavian arteries, singly or combined: by the absence of pressure signs of importance; by the unchanging character and apparently indefinite duration of the affection, the variations being comparatively slight; by the constant coexistence of dilatation of the aorta with eventually secondary insufficiency of the aortic valves; by the difference of treatment required; and lastly, so far as I have at present seen, by its exclusive occurrence in females of a given age—between forty and fifty years, or slightly in excess.*

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* A case answering to the description given will be found in the work of Dr. Grainger Stewart, *Bright's Disease*, 2nd edit., p. 150.

ON SUDDEN DEATH FROM INSUFFICIENCY OF THE AORTIC VALVES.

THAT sudden death may thus be caused is a fact at the present time sufficiently well attested. Though far from common, the result can scarcely be designated as rare, judging from the cases placed on record. The disease stands, as it were, unique in its fatal proclivity. Diseases of the other valves, indeed, may terminate life, but none, as the rule, with such abruptness.* The deaths of one or two prominent members of the profession from this cause, as is alleged, are still fresh in memory.

The event may occur under conditions the most varied; during a state of perfect mental and bodily repose; during one of simple emotional excitement, or during one of ordinary physical exertion. No common precaution, therefore, can guard against the possibility of its occurrence, however remote, where the *well-marked* disease exists.†

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* Death almost equally sudden may occur, but I should suppose most rarely, from fibrinous clots passing from the chambers of the heart and obturating the orifices.

† Apart from this contingency there is, however, a brighter side of the affection. There are cases, rare though they may be, in which compensation remains so perfect as not only to be com-

Opinion is scarcely yet decided as to the proximate cause of death; whether it arises from sudden over-distension of a simply weakened but not necessarily degenerate ventricle, or additionally from some inhibiting influence over the cardiac ganglia. But the concomitant defective supply of blood to the brain, consequent upon the great regurgitation, must be most fully kept in view. In one case mentioned by Vesalius,* the blood contained in the left ventricle amounted to two pounds. Did nervous influence but remain to stimulate one energetic contraction, the ventricle might be relieved of its overcharge, and a recontinuance of cardiac action possibly permitted.

These are the principal factors which enter into the causation of sudden death from aortic insufficiency.

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patible with life, but even with a life of active exertion. From time to time, I see a case of marked aortic insufficiency of many years' duration, and often associated with intercurrent bronchial catarrh. The subject of this affection was, until the last few years, one of the most fearless of steeplechase riders. No day in the field was too long, and no exertion, apparently, too great. I could further select, from my own experience, one or two cases more or less similar. With such we should never attempt in any way to interfere. The sole grounds for treatment are based upon over or under compensation. If, in a case without subjective phenomena, the physical signs of insufficiency are accidentally detected, we should consider well before giving any intimation as to disease, at least to the patient himself, whatever we may do to his friends. Once furnish occasion for consciousness to centre in the belief that something is wrong with the heart, and the chances are that the compensation may soon be ruptured.

* *Vide* Essay by the Author, *On Insufficiency of the Aortic Valves in Connection with Sudden Death, with Notes Historical and Critical,* 1861, p. 24.

Moreover, attention must be paid to the nature, amount of disease, and exact position of the sigmoid valves. Where these are adherent, forming a funnel-shaped diaphragm with only a central chink,* it is difficult to conceive how the coronary arteries could be supplied with blood during ventricular systole; or when, again, large cretaceous masses, or vegetations, extend beyond the origin of these arteries, a similar difficulty occurs. If it were essential that they should be filled during ventricular systole, disturbance, both direct and considerable, might, under the above circumstances, reasonably be expected to follow. But, as this certainly is not the case, it is, perhaps, an indirect argument in favour of their repletion during aortic systole. This view as to the period of repletion is usually ascribed to Brücke.† But the suggestion really originated with Boerhaave.

Pathological results, perhaps, tend more in favour of this view than of the opposing one, on physiological grounds, of Ceradini.

From the very small number of cases of this form of sudden death that have come within my immediate knowledge, the following is selected, inasmuch as the antecedents, both immediate and remote, were known, and the *post-mortem* results carefully noted. The notes were taken, under my supervision, by Mr. W. J. Pickup, at that time one of the resident medical officers at the Royal Free Hospital.*

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* In treating of aortic obstructive disease generally, Professor Germain Sèe (Op. cit. p. 5) remarks: 'This lesion appears to become daily more rare, and the more it is sought for, the less the likelihood of its being found. It occurs, however, in old subjects; but it is exceptional in children, and extremely rare in adults.' This opinion appears to be supported both by the clinical and pathological experience of one of the very ablest among our own observers. Reynolds' *System of Medicine*, vol. 4, p. 611.

† Michael Foster, A.M., M.D., F.R.S., *A Text Book of Physiology*, 1877, p. 104.

E. P——, aged thirty, housemaid, unmarried, was admitted in the early part of October 1876. During the last eight years she had been more or less frequently under the care of Dr. Cockle. Her illness originated in a severe attack of rheumatic fever, during the progress of which the heart became implicated; both aortic and mitral valves were affected, the former, however, to a much greater extent, and leaving as a permanent result free regurgitation through the aortic orifice. Dilated hypertrophy of the left ventricle gradually supervened. In course of time, however, so perfect a compensation was established that she was enabled to resume her occupation, remaining in her last situation upwards of three years. In the spring of last year she was admitted with a second attack of rheumatic fever. Although protracted, this attack, so far as could be ascertained, produced no further mischief in the heart, unless it were to render the slight mitral murmur left by the first attack a little more manifest, but the characteristic murmur of aortic reflux was always strongly marked.

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* Hospital Reports, *Lancet*, Jan. 20th, 1877.

When sufficiently recovered she was removed to a convalescent institution, where she remained some weeks. Shortly after her return, she was re-admitted into the hospital in a state of alarming debility. Her complexion was of waxy paleness; her breathing hurried; the pulse less bounding; the cardiac impulse diminished;* the lower extremities were thickly covered with dark, purpuric spots. After some days perfect rest in bed, good diet, and ferruginous tonics, the purpuric spots gradually lost their dark hue, and the complexion assumed, to some extent, a healthier aspect. On the morning of the day of her death—Oct. 13th—she looked and said she felt better, and seemed to be progressing favourably. She had no untoward symptoms during the greater part of the day. Five minutes before she died Mr. Pickup passed her bedside, when she was sleeping quietly on her left side. Immediately afterwards he was called back to the ward and informed that, just after he left, she awoke, screamed, threw up her arms, and was dead before the nurse could reach the bedside. She was now greatly cyanosed, and the temperature taken at once was 102·4 deg., though on the previous day it had been scarcely above normal.

Autopsy, twenty-four hours after death.—Both lungs were considerably congested. The left ventricle was

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* Essay cited, p. 25.

distended and filled with clotted blood, the clot extending up into the aorta. The aortic semilunar valves were thickened, cord-like, and rigid, preventing closure, and admitting free regurgitation. The orifices of the coronary arteries were patent. Patches of atheroma existed in the aorta. The segments of the mitral valves were thickened and fringed with vegetations, but capable of closure; two threads from these, an inch in length, hung free in the cavity of the ventricle. The walls of the left ventricle were thickened, the chamber immensely dilated. The right ventricle was also much dilated. No marked evidence of either fatty or fibroid degeneration was observable on microscopic examination. The heart, emptied of blood, weighed sixteen ounces. There was nothing special to note in the other organs beyond a slight adhesion of old-standing between the pericardium and the lower and anterior surface of the left ventricle. The sac contained about an ounce of clear serum.

REMARKS BY DR. COCKLE.—This case exhibits a comparatively rare though direct result of aortic insufficiency—sudden death—in its nearly purest form; for the mitral lesion may, practically, be disregarded. The valvular incompetence resulting from the first attack of rheumatic fever caused dilatation and consecutive hypertrophy of the walls of the left ventricle. So perfect an adjustment was gradually effected, that the working of the heart was fairly well maintained for a series of years. A second attack of rheumatic fever was followed by great deterioration of the general health and obvious loss of power; the cardiac adjustment now failed, and eventually, as happens in such cases, at some quite unexpected moment, sudden cessation of the contractile power of the ventricle ensues when its cavity is distended with refluent blood during diastole. In the case detailed, sleep might have had some indirect influence. The enlargement of the right ventricle finds ready explanation.

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SHORTLY WILL BE PUBLISHED,

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IN

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